Pulmonary artery banding in isolated or complicated ventricular septal defects
Results and effects on growth

Samuel Menahem[1] and A. W. Venables

From the Department of Cardiology, Royal Children's Hospital,
Flemington Road, Parkville, Victoria, Australia

Experience with patients undergoing pulmonary artery banding for control of substantial left-to-right shunt associated with large ventricular septal defects has been reviewed in order to assess the effects of banding and the growth patterns of the subjects in relation to changes in cardiac loading.

The series covers a period from 1960 through 1968. All 22 infants studied had large ventricular septal defects with increased arterial pressures and substantial left-to-right shunts. About 50 per cent of the patients had associated cardiac anomalies; 7 had persistence of the ductus arteriosus and 4 coarctation of the aorta. These associated anomalies where possible were dealt with at the time of or before the banding procedure. Generally the band was applied to the limit of tolerance.

There were 6 surgical deaths which included 3 patients with substantial associated anomalies. Nine patients had a satisfactory immediate response, 1 had a partial response, while 6 failed to improve after operation. Repeat study in the latter group showed persisting left-to-right shunt despite the band being in position in all except 1 subject in whom the band had cut through the pulmonary artery. Three patients were rebanded, with 1 survivor who was satisfactory. The remaining 3 patients were managed medically, with 2 survivors ultimately improving some months later.

The growth of the whole group was retarded. Most, in addition, had evidence of intrauterine growth retardation. Cardiac failure was associated with minimal or no growth. After effective banding had controlled the large left-to-right shunt, improvement in growth was generally seen, some patients in addition displaying catch-up growth. Thus, resumption or acceleration of growth was a further criterion of satisfactory banding.

Large ventricular septal defects still cause formidable morbidity and appreciable mortality during infancy (Morgan, Griffiths, and Blumenthal, 1960; Ritter *et al.*, 1965; Nadas, 1967; Hoffman, 1968). Pulmonary artery banding in such patients can control the large left-to-right intraventricular shunts which lead to cardiac failure. Survival and clinical improvement may thereby be enhanced (Muller and Dammann, 1952).

This paper reports experience at the Royal Children's Hospital, Melbourne, paying particular attention to two main aspects: namely, failure to produce improvement in certain patients after the procedure, and the growth of the patients before and after banding.

Subjects and methods

During the period 1960 through 1968 pulmonary artery banding was performed in 22 infants with large ventricular septal defects with or without associated anomalies (Table 1). There was an overall mortality of about 40 per cent (Tables 2 and 3). All patients presented early with the main symptoms of dyspnoea or failure to thrive. Varying degrees of cardiac failure were noted, two-thirds of the infants requiring prolonged feeding by gastric tube in addition to intensive medical therapy.

Catheter studies showed a ratio of pulmonary to systemic flow of greater than 2·0 in all subjects (Kirklin, 1965). Nineteen of the 22 patients had equalized pulmonary and systemic systolic pressures, while the remaining 3 had moderate pulmonary hypertension.

The associated anomalies are noted in Table 1. Five patients had a rise in oxygen saturation at right atrial level in the initial catheter study. In 2 this was not detected at subsequent follow-up

TABLE I   *Associated cardiac anomalies*

Anomalies	Numbers
'Isolated' ventricular septal defect	15*
Ventricular septal defect and preductal coarctation of aorta	2
Ventricular septal defect and simple coarctation of aorta	2
Ventricular septal defect and atrial rise in oxygen saturation	2
Ventricular septal defect, persistent ductus arteriosus, and unroofed coronary sinus	I
Total	22

* Includes 2 subjects with initial atrial rise in oxygen saturation not detected later; 1 subject with spontaneous closure of duct at term; 2 subjects with small ducts closed at time of banding; 1 subject with small duct noted at necropsy; 1 subject with bicuspid aortic valve (see text).

study. Another was shown to have an unroofed coronary sinus. One patient died without necropsy while the fifth awaits restudy.

There were 4 infants with a persistent ductus arteriosus. One premature infant had spontaneous closure of the duct at a time corresponding to full-term gestation. Another had a small duct diagnosed at necropsy. The remaining 2 patients had their duct diagnosed only by the passage of a catheter through the duct. The ducts were ligated at the time of banding.

There were 2 patients with preductal coarctation of the aorta. The ducts were closed before or at the time of banding but only 1 patient had resection of the coarctation. There were 2 other infants with coarctation of the aorta which was resected at the time of banding.

The indications for pulmonary artery banding in infancy and the technique of the procedure were essentially similar to other workers (Nadas, 1967; Albert *et al.*, 1961; Morrow and Braunwald, 1961; Goldblatt *et al.*, 1965; Idriss, Riker, and Paul, 1968). The bands employed were of various materials. Braided silk, and nylon or Teflon ribbon or crimped Teflon secured by suturing, were used. The external diameter of the pulmonary artery was narrowed to approximately one-third to one-half of its original size, usually aided by measurement of right ventricular and peripheral pulmonary arterial pressure. The final degree of narrowing was often just short of that which caused cardiac dilatation and arrest.

Banding was most frequently performed at about 3 months of age, the range being 3 weeks to 7 months. Follow-up was from 3 to 9 years.

Individual growth curves were drawn for each subject using the Boston Growth Charts (Stuart, 1939; Stuart and Reed, 1951). The height and weight percentiles of the survivors were noted at selected ages. The relation of the birthweight to gestational age of each subject was assessed (Kitchen, 1968). Bone age measurements were made at varying ages (Greulich and Pyle, 1959). The heights and weights of respective parents and sibs were also obtained.

TABLE 2   *Result of initial pulmonary artery banding*

Abnormality	Response				
	Satisfactory	Partial	Inadequate	Death	Total
Ventricular septal defect	8	I*	3	3	15
Ventricular septal defect and preductal coarctation			2†		
Ventricular septal defect and simple coarctation of aorta			I	I	2
Ventricular septal defect and 'A.S.D.'	I			I	2
Ventricular septal defect + persistent ductus arteriosus + unroofed coronary sinus				I	I
Total	9	I	6	6	22

* Subsequently developed microcephaly and died.
† 1 subject with unresected coarctation.
'A.S.D'. = rise in oxygen saturation as yet unexplained.

Results

The results of pulmonary artery banding are shown in Table 2. In 9 patients, 8 of whom had isolated ventricular septal defects, there was a satisfactory response. An immediate overall clinical improvement occurred with a decrease in the infants' dyspnoea, return of ability to feed normally, and early discharge from hospital.

There were 6 deaths at or shortly after operation, ventricular fibrillation occurring in one patient as the chest incision was made. Of these 6 patients, 3 had substantial associated cardiac anomalies.

One infant had partial improvement in his clinical state. He subsequently developed microcephaly and died at about 6 months of age.

Six infants had an inadequate response in spite of a similar technique. The outcome is noted in Table 3. These patients showed little improvement and required continuation of

TABLE 3   *Outcome of subjects with inadequate response to initial surgery*

Management	Response		
	Early improvement	Late improvement	Death
Medical therapy only		2	I*
Further surgery:			
Reband			I
Reband with repeat resection of coarctation	I		
Reband without resection of coarctation			I
Total	I	2	3

* Band had cut through pulmonary artery (see text).

intensive medical therapy. When restudied all showed ratios of pulmonary to systemic flow still greater than 2·0, with peripheral pulmonary arterial hypertension. Angiocardiography or withdrawal tracings confirmed the presence of a constricting band as shown in Fig. 1, except in 1 patient where the band was noted to lie within the lumen of the pulmonary artery (Idriss *et al.*, 1968). Necropsy showed the band to have cut through the lower part of the pulmonary artery, the defect so formed being filled by the upper surface of the left atrium. The 2 infants, who had medical therapy and survived (Table 3), followed a stormy course complicated by repeated respiratory infections before improvement was noted 2 months and 5½ months later (Case 10 – angiogram seen in Fig. 1 and Case 3, respectively).

The onset of cyanosis was observed by the age of 2 years in 5 patients. Two others have failed to develop cyanosis; one (Case 3) now aged 5½ years who still has evidence of left-to-right shunting on recent study, and the other (Case 12) who was shown on repeat catheterization at the age of 6 years to have had spontaneous closure of his ventricular septal defect (Nghiem, Harris, and Tyson, 1969; Subramanian and Wagner, 1970).

Gross growth retardation was shown in all infants. This retardation was present to some extent at birth. There were 12 infants of low birthweight (Gruenwald, 1965) when related to gestational age (Fig. 2). Weight and to a lesser extent length were depressed when the infants presented (Fig. 3). By the age of 3 months the growth retardation was gross. Some improvement was noted as early childhood was reached, being more conspicuous in weight than in height (Fig. 4). The above figures are statistically significant on analysis, a summary of this analysis being noted in Table 4.

There was no significant correlation between subjects' height and weight with that of their respective parents or sibs.

The longitudinal growth of those patients who succumbed is seen in Fig. 5 and 6. The survivors showed varying responses to pulmonary artery banding as exemplified by Fig. 7, 8, and 9.

FIG. 1 *Angiogram showing constricting pulmonary artery band in position in Case 10 with inadequate response.*

TABLE 4 *Summary of statistical analysis of growth data*

Figure		Arbitrary percentile division	Number below division	Number above division	Test used	Result
1	Birthweight to gestational age	25	12	10	χ^2*	P = <0·01
2	Height on presentation	3	7	5	Cumulative binomial distribution†	P = <0·01
	Weight on presentation	3	16	6	Cumulative binomial distribution	P = <0·01
3	Height at 3 mth	3	10	5	Cumulative binomial distribution	P = <0·01
	Height at 12 mth	3	7	5	Cumulative binomial distribution	P = <0·01
	Height at 2 yr	3	5	7	Cumulative binomial distribution	P = <0·01
	Height at 6 yr	10	4	2	Cumulative binomial distribution	P = <0·01
	Weight at 3 mth	3	19	0	Cumulative binomial distribution	P = <0·01
	Weight at 12 mth	3	10	2	Cumulative binomial distribution	P = <0·01
	Weight at 2 yr	3	6	6	Cumulative binomial distribution	P = <0·01
	Weight at 6 yr	3	2	4	Cumulative binomial distribution	P = <0·01

Arbitrary division at 25th percentile, then expected frequency below and above division is 22/4 and 22 × 3/4 respectively, 1 degree of freedom, chi-square = 10·24, P = <0·01.
Arbitrary division at 3rd percentile, then P ('success') = P (below 3%) = 0·03. Using the cumulative binomial distribution, the probability of having at least 7 'successes' in a total of 12 subjects or 'trials' = 0·01 (Eilon, 1962).
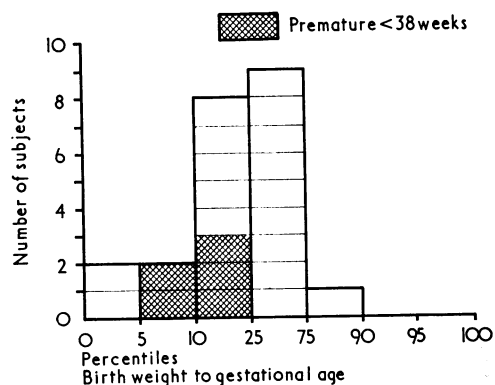
FIG. 2 *Relation of patients' birthweight to their gestational age utilizing percentile ratings.*

In a few patients impairment of growth was noted after the second year, coinciding with the onset of increasing clinical cyanosis.

Bone age was retarded after the age of 6 months in 10 of the 12 survivors in whom it was estimated.

Discussion

The potential effectiveness of pulmonary artery banding in controlling large intraventricular left-to-right shunts has been well documented (Nadas, 1967; Morrow and Braunwald, 1961; Goldblatt *et al.*, 1965). Banding may transform a sick, pale, dyspnoeic infant to one who is relatively well and able to feed orally. This result encourages further
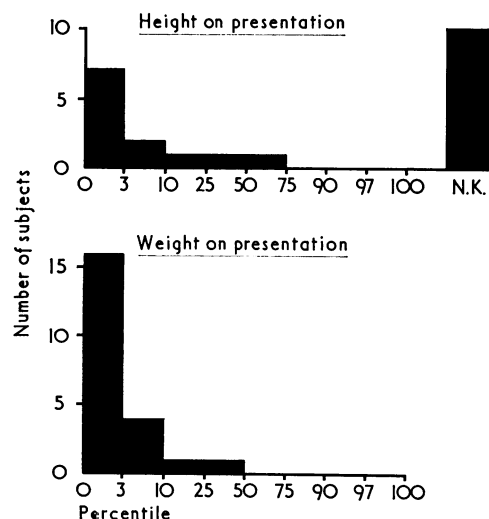
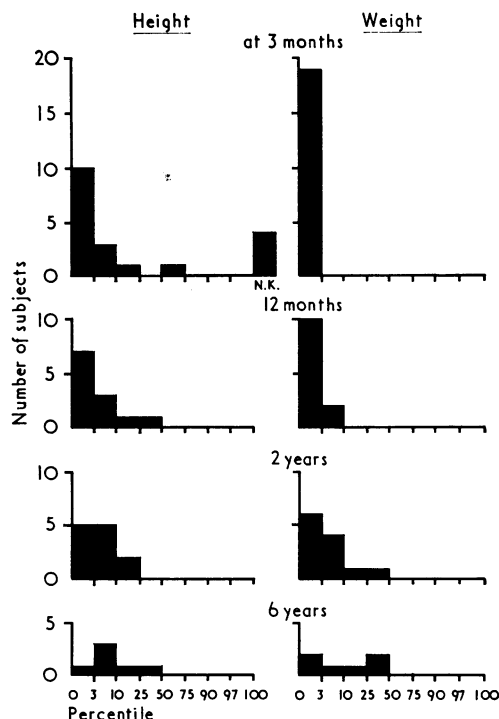FIG. 3 *Height and weight percentiles of all patients at presentation. (N.K. = not known).*

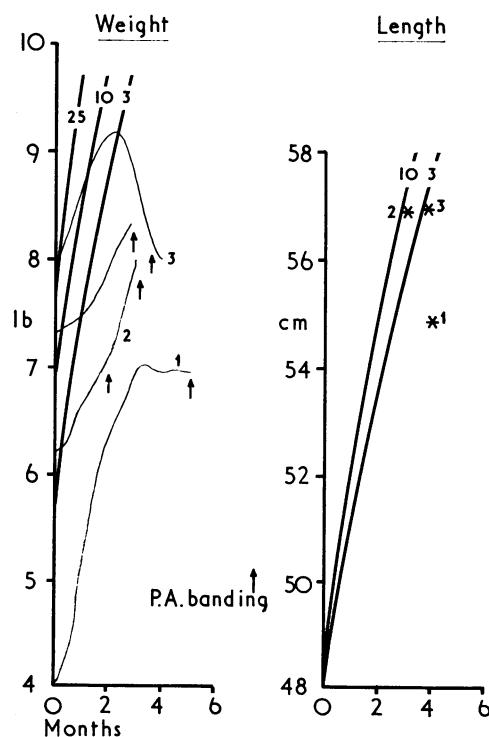FIG. 4 *Height and weight percentiles of survivors at selected ages (N.K. = not known).*

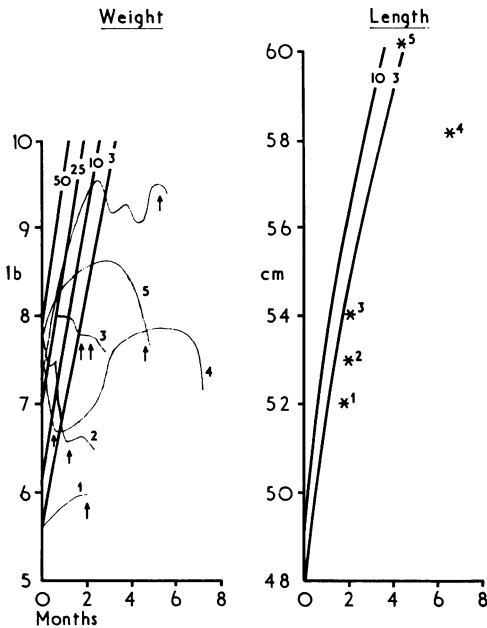FIG. 5 *Growth curves of female infants who died at or soon after initial operation.*

FIG. 6 *Growth curves of male infants who died at or soon after initial operation. Curve labelled 4 relates to a subject who developed microcephaly and died at 6 months.*

use of this procedure despite its initial and later hazards. Early surgical deaths often reflect the poor clinical condition of the infant at the time of banding, though the presence of various associated cardiovascular anomalies
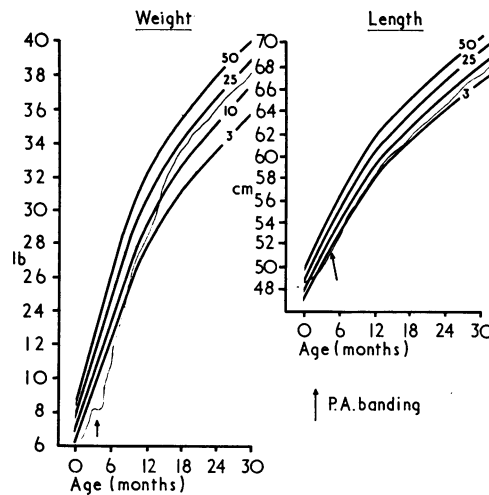

FIG. 8 *Growth curve of a patient who showed catch-up growth after effective pulmonary artery banding.*

makes a significant contribution to prognosis (Goldblatt *et al.*, 1965).

Failure to obtain immediate improvement despite the employment of an apparently adequate technique at operation has been reported briefly in a number of papers (Albert *et al.*, 1961; Goldblatt *et al.*, 1965; Subramanian and Wagner, 1970; Goldberg *et al.*, 1966; Smith *et al.*, 1966; Stark *et al.*, 1969).
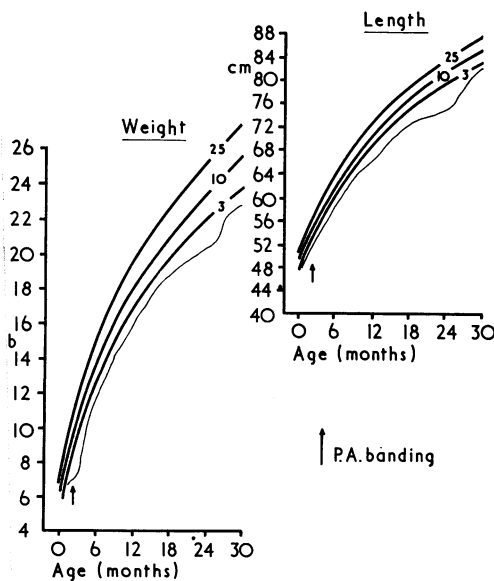

FIG. 7 *Growth curve of a patient who showed no catch-up growth after effective pulmonary artery banding.*
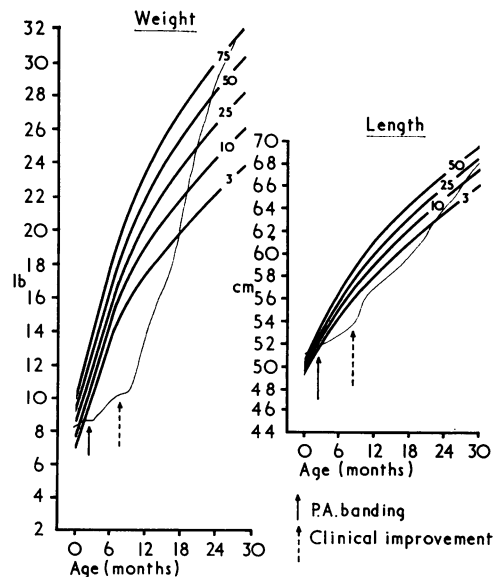

FIG. 9 *Growth curve of a subject who after unsatisfactory pulmonary artery banding showed improvement in growth only after clinical improvement.*

Though this may mean that the band is in fact not tight enough to achieve its purpose, this may occur when at operation the band has been applied as tightly as the heart will tolerate. It is important to recognize that the banding may prove inadequate even in such circumstances. The present study has shown that in such patients with an inadequate response, improvement may occur over a few months even without further operation as the constricting band becomes relatively more effective in controlling the shunt, without decrease in size of the defect or rise in pulmonary vascular resistance. However, during this critical period of inadequate control of the large shunt the infant remains unwell and at the same risk as before banding. He continues to require intensive medical therapy, and death may occur, often precipitated by an intercurrent respiratory infection.

In this study the patients who failed to improve despite apparently adequate banding at the time of operation were shown on repeat study to have persisting large left-to-right shunts with peripheral pulmonary artery hypertension, the constricting band being in position except in the one case where it had cut through the pulmonary artery. In contrast, an infant who had an excellent response to banding had on repeat study a relatively normal peripheral pulmonary artery pressure, with only slight increase in pulmonary flow.

Further banding was performed in 3 patients who had had an initial unsatisfactory response, the 1 survivor, who also had repeat resection of her coarctation, showing conspicuous improvement. Two others, who were not rebanded, remained unwell and in cardiac failure until clinical improvement occurred.

Growth retardation has been previously noted in infants with large left-to-right shunts (Mehrizi and Drash, 1962; Maxwell, Wurfel, and Burnell, 1966; Umansky and Hauck, 1962). This study further illustrates the severity of the growth retardation, the infants with cardiac failure deviating progressively from the normal lines of growth despite apparently adequate intake. Effective pulmonary artery banding was associated with restarting or acceleration of growth, especially with respect to weight (Morrow and Braunwald, 1961; Craig and Sirak, 1963; Goldblatt et al., 1965; Idriss et al., 1968). However, subsequent growth showed two patterns of response once haemodynamic control was achieved. Four infants showed little, if any, catch-up growth (Prader, Tanner, and von Harnack, 1963), either in height or weight. Their subsequent growth curves followed the percentile line to which they had regressed before effective banding (Fig. 7). All these subjects were infants whose birthweight was low when related to their gestational age (Mitchell, Berendes, and Clark, 1967; Menahem, 1971). In addition, 3 out of the 4 were premature on dates (Table 5). In contrast, in 5 other patients growth not only started again, with effective haemodynamic control, but subsequently accelerated, and crossing of percentile lines occurred both in height and in weight, indicating catch-up growth (Fig. 8) (Albert et al., 1961). The remaining three patients showed catch-up growth with respect to weight, but not height.

TABLE 5 *Summary of follow-up of growth data*

Case No.	Sex	Follow-up	Pregnancy	Gestational age	Birth-weight age	Gain in height channel	Age of occurrence	Present height channel	Gain in weight channel	Age of occurrence	Present weight channel
1	F	7 yr	Normal	Full term	Normal	No		25–50%†	Yes	11 mth	10–25%†
2	M	6 yr	Normal	Full term	Normal	No		3–10%	Yes	13 mth	25–50%
3*	F	5 yr	Virus at 6 wk	Full term	Normal	Yes	22 mth	25–50%	Yes	18 mth	50–75%
4	F	5 yr	Normal	Full term	Low	No		<3%	Yes	38 mth	3–10%
5	M	6 yr	Normal	Full term	Low	Yes	6 mth	3–10%‡	Yes	25 mth	10–25%
6	M	7 yr	Normal	Full term	Low	Yes	8 mth	3–10%	Yes	8 mth	10–25%
7	F	6 yr	'Drugs'	Full term	Low	Yes	7 mth	<3%‡	Yes	16 mth	<3%‡
8	M	4 yr	Excess alcohol, ante-partum haemorrhage	Full term	Very low	No		<3%	No		<3%
9	F	3 yr	Toxaemia	36 wk	Low	No		<3%	No		<3%
10*	F	6 yr	Normal	33 wk	Low	Yes	18 mth	3–10%‡	Yes	24 mth	<3%‡
11	M	4 yr	Toxaemia, twins	37 wk	Low	No		<3%	No		<3%
12	M	9 yr	Toxaemia	35 wk	Very low	No		<3%	No		<3%

* Had delay in haemodynamic control.
† After debanding.
‡ Subsequent loss in channel.
Present = 1970.

In the cases where banding did not produce control of the intracardiac shunt, improvement in growth did not start until clinical improvement occurred (Fig. 9).

Retardation in skeletal maturity may simply reflect the overall growth retardation (Ryba, Hedvall, and Carlgren, 1967). It may also indicate potential for subsequent improvement of growth which will only be manifest by follow-up of these children into teenage and adult life (Bayer and Robinson, 1969).

Some understanding of the above growth patterns may be obtained by recent work performed to elucidate the basis for the growth retardation (Cheek, 1968). Haemodynamic disturbances, especially if severe as in these infants, may be important through such mechanisms as cardiac failure, 'hypermetabolism' (Lees *et al.*, 1965), repeated respiratory infection, hypoxia, acidosis, inadequate intake or excess loss (Neill, 1968). These mechanisms are thought not only to lead to growth retardation at the time of their action, but may also have a prolonged effect by inhibition of cellular division and cellular size so that even when correction or control of the haemodynamic disturbance occurs, incomplete recovery is noted (Cheek, Graystone, and Mehrizi, 1966; Naeye, 1965). The earlier the 'insult' in relation to the development of the infant the more severe the outcome appears to be, as seen in those infants who were premature. Possible poor intrinsic growth potential is suggested by the intrauterine growth retardation present in some patients. This potential may then become of major importance when the haemodynamic disturbance is controlled (Umansky and Hauck, 1962; Menahem, 1971). This intrauterine growth retardation may be attributable, together with the cardiac abnormalities, to a yet unknown common cause (Cheek, 1968).

The onset of increasing hypoxia may have contributed to late fall-off in growth in those subjects in whom cyanosis was noted.

Conclusion

The criteria of successful banding should not be limited to operative survival and ultimate recovery. They should also include a rapid satisfactory response enabling normal feeding and prompt discharge from hospital. Application of the pulmonary artery band even to the limit of cardiac tolerance, together with correction of associated anomalies, did not guarantee such a response in the present series. Failure to obtain a satisfactory response after banding should encourage further early investigation to assess the status of the band and the continuing haemodynamic disturbance. Repeat banding may need to be considered together with surgical correction of any residual associated anomalies that may be contributing to the haemodynamic picture.

The poor growth of the infants with large left-to-right intracardiac shunts in this series appears to be an indication of the severity of the lesion. This study shows that resumption of growth provides a good index of control of shunt after pulmonary artery banding even in the absence of catch-up growth and return to normal channels. When the response to banding was delayed, the resumption of growth was also delayed, the start coinciding with clinical improvement. Failure of occurrence of normal growth, even with control of the haemodynamic disturbance, may be related to intrauterine growth retardation in addition to the long-term effects of the period of severe postnatal cardiac failure.

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